LAW OFFICES
ABER, GOLDLUST, BAKER & OVER
(AN ASSOCIATION OF LAW PRACTICES)
702 KING STREET, SUITE 600
P.O. BOX 1675
WILMINGTON, DELAWARE 19899-1675

GARY W. ABER, P.C.
PERRY F. GOLDLUST, P.A.*
DARRELL J. BAKER, P.A.
SUSAN C. OVER, P.C.
SHAUNA T. HAGAN
SAAGAR B. SHAH

*ALSO ADMITTED IN NEW YORK
**ALSO ADMITTED IN PENNSYLVANIA

(302) 472-4900
TELECOPIER (302) 472-4920

June 2, 2006

The Honorable Kent A. Jordan
United States District Court
844 King Street
Wilmington, DE 19801

RE: Hawksley v. Unum Life Insurance Company of America, et. al.
C.A. No.: 04-1305

Dear Judge Jordan:

This is the Plaintiff's Status Report as required by the Scheduling Order in the above-captioned matter.

The plaintiff has informally attempted to suggest discovery, and to schedule depositions in this matter with defendant's counsel. (See Letter: Aber to Wolgenmutch: 5/3/06, attached hereto as Exhibit "A"). In response to that request, the defendant has erroneously written back that the discovery requests were untimely (Letter: Wolgenmutch to Aber: 5/22/06, Exhibit "B"). The discovery requests were timely, since the discovery cut off in this matter is July 25, 2006 (Dk-20).

The plaintiff's request for depositions of the defendant's in-house physicians is appropriate. Unlike Semien v. Life Insurance Co of North America, 436 F.3d 805 (2006) the plaintiff is entitled to discovery. In Semien the Court upheld the denial of discovery of **independent** physicians hired by the plan administrator. There, the Court, in upholding the denial of discovery stated:

> "These physicians were not employees of the company, and they did not fail to analyze relevant medical evidence..." Semien, supra, at p. 814.

The crux of this matter is, whether or not the in-house physicians of the defendant did properly analyze medical evidence. The claim of disability by the plaintiff, was that she suffered from intractable nausea, which resisted treatment, and prevented her from continued employment. The plaintiff did have radiological confirmation of a brain tumor. However, the defendant's in-house physicians stated that the location of the tumor would not cause such intractable nausea. This is contrary to established medical literature (Exhibit "C").

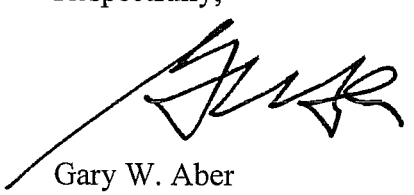
In Medford v. Metropolitan Life Insurance Co., 244 F.Supp.2d 1120 (D.Nev. 2003) the Court allowed discovery of the physicians, who offered opinions on behalf of the employer's insurance company, in order to determine the independence or neutrality of those physicians, and the medical opinions relied upon the defendants. See also: Waggear v. Unum Life Insurance Co. of America, 238 F.Supp.2d 1179 (S.D. Cal. 2002)(holding that such discovery was appropriate in order to determine the degree of conflict of interest of the plan's administrator).

With regards to the 30(b)(6) deposition suggested to the defendant, in reviewing the defendant's letter to the Court dated May 30, 2006, I am unclear as exactly to what Unum states that it will stipulate. I have attempted to contact defendant's counsel, after receiving that letter, but he was out of the office until June 5, 2006, and I hope to clarify that matter prior to the telephone conference with the Court.

As to the 30(b)(6) deposition as to how the claim was handled, whether any determination as to how and what materials in the administrative filed were actually reviewed, and considered, would go to a violation of the applicable standard for review by Unum. Thus, such discovery would be appropriate.

Thank you for Your Honor's consideration.

Respectfully,



Gary W. Aber

GWA/mac
cc: Kirk Wolgenmutch, Esquire

EXHIBIT A

May 3, 2006

Kirk Wolgenmutch, Esquire
Stevens & Lee
1105 N. Market Street, 7th Floor
Wilmington, DE 19801

RE: Hawksley v. Unum Life Insurance Company of America, et. al.

Dear Kirk:

I would like to schedule some depositions in this matter. The two individuals I would like to depose are Lani Graham, M.D., listed as the reviewer on October 21, 2003, and Alan Neuren, M.D., based on a review dated July 3, 2003.

I would also like to do two 30(b)(6) depositions. First would be of a person who could testify as to the relationship between the employer and Unum, in order to establish a standard of review by the Courts. Alternatively, perhaps we could stipulate as to what that standard is. Also, I would like to do a 30(b)(6) deposition of the most knowledgeable person in the handling and administration of this claim, the sequence of events, and how decisions were arrived at.

Please give me a call so we can discuss scheduling these matters.

Yours very truly,

Gary W. Aber

GWA/mac

EXHIBIT B

STEVENS & LEE
LAWYERS & CONSULTANTS

111 North 6th Street
P.O. Box 679
Reading, PA 19603-0679
(610) 478-2000 Fax (610) 376-5610
www.stevenslee.com

Direct Dial: (610) 478-2240
Email: klw@stevenslee.com
Direct Fax: (610) 988-0810

May 22, 2006

Gary W. Aber, Esquire
Aber, Goldlust, Baker & Over
702 King Street
P.O. Box 1675
Wilmington, DE 19899-1675

Re: *Hawksley v. UNUM Life Insurance Company of America*

Dear Gary:

I am in receipt of your letter dated May 3, 2006 wanting to schedule depositions in the above-referenced matter. As I am sure you are aware, discovery in this matter concluded on January 25, 2006. Accordingly, I believe your discovery requests are untimely and, because this is an ERISA matter, unnecessary because discovery is not appropriate where the Court must make its findings on the administrative record. If you would like to discuss this matter, please do not hesitate to contact me.

Very truly yours,

STEVENS & LEE

Kirk L. Wolgemuth

KLW:emvl

EXHIBIT C

14TH EDITION

Harrison's

PRINCIPLES of INTERNAL MEDICINE

EDITORS

Anthony S. Fauci, MD
Chief, Laboratory of Immunoregulation; Director, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda

Eugene Braunwald, AB, MD, MA (Hon), MD (Hon), ScD (Hon)
Distinguished Hersey Professor of Medicine, Faculty Dean for Academic Programs at Brigham and Women's Hospital and Massachusetts General Hospital, Harvard Medical School; Vice-President for Academic Programs, Partners HealthCare System, Boston

Kurt J. Isselbacher, AB, MD
Mallinckrodt Professor of Medicine, Harvard Medical School; Physician and Director, Massachusetts General Hospital Cancer Center, Boston

Jean D. Wilson, MD
Charles Cameron Sprague Distinguished Chair and Clinical Professor of Internal Medicine, The University of Texas Southwestern Medical Center, Dallas

Joseph B. Martin, MD, PhD, FRCP (C), MA (Hon)
Dean of the Faculty of Medicine; Caroline Shields Walker Professor of Neurobiology and Clinical Neuroscience, Harvard Medical School, Boston

Dennis L. Kasper, MD, MA (Hon)
William Ellery Channing Professor of Medicine, Harvard Medical School; Director, Channing Laboratory; Co-Director, Division of Infectious Diseases; Executive Vice-Chairman, Department of Medicine, Brigham and Women's Hospital, Boston

Stephen L. Hauser, MD
Chairman and Betty Anker Fife Professor, Department of Neurology, University of California San Francisco, San Francisco

Dan L. Longo, AB, MD, FACP
Scientific Director, National Institute on Aging, National Institutes of Health, Gerontology Research Center, Bethesda and Baltimore

McGraw-Hill
HEALTH PROFESSIONS DIVISION

New York St. Louis San Francisco Auckland Bogotá Caracas Lisbon London Madrid
Mexico City Milan Montreal New Delhi San Juan Singapore Sydney Tokyo Toronto

has important implications for therapy. The picture of complete basilar insufficiency, however, is easy to recognize. A combination of bilateral long tract signs (sensory and motor) with signs of cranial nerve and cerebellar dysfunction suggests this diagnosis. A "locked-in" state of e quadriplegia, bifacial and oropharyngeal palsy, and paralysis orizontal gaze occurs with bilateral basis pontis infarction. Stupor indicates dysfunction of the reticular activating system, and when combined with third cranial nerve palsies it suggests devastating upper midbrain infarction (see Chap. 24). The therapeutic goal, however, is to recognize *impending* basilar occlusion before devastating infarction occurs. A series of TIAs or a slowly progressive, fluctuating stroke are extremely significant as they often herald an atherothrombotic occlusion of the distal vertebral or proximal basilar artery.

TIAs in the proximal basilar distribution may produce dizziness (often described by patients as "swimming," "swaying," "moving," "unsteadiness" or "light-headedness"). Other symptoms that warn of basilar thrombosis include diplopia, dysarthria, facial or circumoral numbness, and hemisensory symptoms. In general, symptoms of basilar branch TIAs affect one side of the brainstem, whereas symptoms of basilar artery TIAs usually affect both sides, though a "herald" hemiparesis has been emphasized as an initial symptom of basilar occlusion. Most often TIAs, whether due to impending occlusion of the basilar artery or a basilar branch, are short-lived (5 to 30 min) and repetitive, occurring several times a day. The pattern suggests intermittent reduction of flow.

Atherothrombotic occlusion of the basilar artery with *brainstem infarction* usually causes *bilateral* brainstem signs. For ex , gaze paresis or internuclear ophthalmoplegia may be associated with ipsilateral hemiparesis. By contrast, occlusion of a branch of the basilar artery usually causes *unilateral* symptoms and signs involving motor, sensory, and cranial nerves.

SUPERIOR CEREBELLAR ARTERY Occlusion of this artery results in severe ipsilateral cerebellar ataxia, nausea and vomiting, dysarthria, and contralateral loss of pain and temperature sensation over the extremities, body, and face. Partial deafness, ataxic tremor of the ipsilateral upper extremity, Horner's syndrome, and palatal myoclonus may also occur. Partial syndromes are common (Fig. 366-8).

ANTERIOR INFERIOR CEREBELLAR ARTERY Occlusion produces variable degrees of infarction because the size of this artery and the territory it supplies vary inversely with those of the posterior inferior cerebellar artery. The principal symptoms include ipsilateral deafness, facial weakness, true vertigo (whirling dizziness), nausea and vomiting, nystagmus, tinnitus, cerebellar ataxia, Horner's syndrome, and paresis of conjugate lateral gaze. The opposite side of the body loses pain and temperature sensation. An occlusion close to the origin artery may cause corticospinal tract s (see Fig. 366-10).

Occlusion of one of the five to seven short circumferential branches of the basilar artery affects the lateral two-thirds of the pons and middle or superior cerebell

duncle, whereas occlusion of one of the 7 to 10 paramedian branches affects a wedge-shaped area on either side of the medial pons (Figs. 366-8 to 366-10).

Lacunar Disease The term *lacunar infarction* refers to infarction following atherothrombotic or lipohyalinotic occlusion of one of the penetrating branches of the circle of Willis, middle cerebral artery stem, or vertebral and basilar arteries.

Pathophysiology The middle cerebral artery stem, the arteries comprising the circle of Willis (A1 segment of the anterior cerebral artery, anterior and posterior communicating arteries, precommunal segment of the posterior cerebral arteries), and the basilar and vertebral

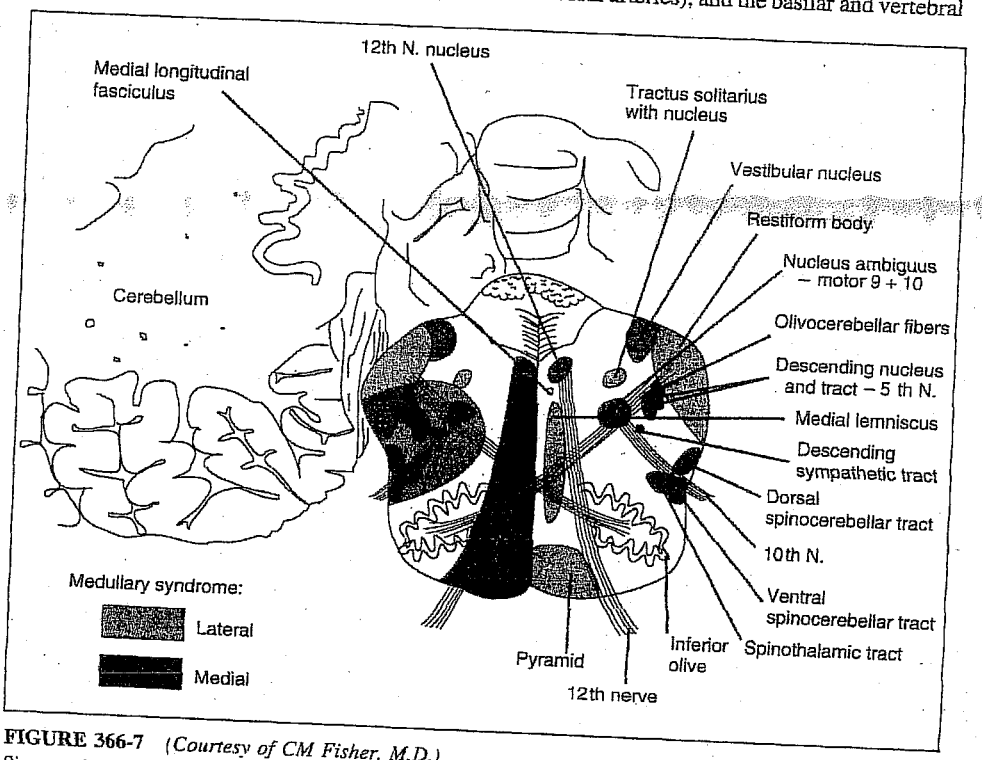


FIGURE 366-7 *(Courtesy of CM Fisher, M.D.)*
Signs and symptoms: *Structures involved*

1. Medial medullary syndrome (occlusion of vertebral artery or of branch of vertebral or lower basilar artery)
   On side of lesion
   Paralysis with atrophy of half the tongue: *Ipsilateral twelfth nerve*
   On side opposite lesion
   Paralysis of arm and leg sparing face; impaired tactile and proprioceptive sense over half the body: *Contralateral pyramidal tract and medial lemniscus*
2. Lateral medullary syndrome (occlusion of any of five vessels may be responsible—vertebral, posterior inferior cerebellar, superior, middle, or inferior lateral medullary arteries)
   On side of lesion
   Pain, numbness, impaired sensation over half the face: *Descending tract and nucleus fifth nerve*
   Ataxia of limbs, falling to side of lesion: *Uncertain—restiform body, cerebellar hemisphere, cerebellar fibers, spinocerebellar tract (?)*
   Nystagmus, diplopia, oscillopsia, vertigo, nausea, vomiting: *Vestibular nucleus*
   Horner's syndrome (miosis, ptosis, decreased sweating): *Descending sympathetic tract*
   Dysphagia, hoarseness, paralysis of palate, paralysis of vocal cord, diminished gag reflex: *Issuing fibers ninth and tenth nerves*
   Loss of taste: *Nucleus and tractus solitarius*
   Numbness of ipsilateral arm, trunk, or leg: *Cuneate and gracile nuclei*
   On side opposite lesion
   Impaired pain and thermal sense over half the body, sometimes face: *Spinothalamic tract*
3. Total unilateral medullary syndrome (occlusion of vertebral artery): Combination of medial and lateral syndromes
4. Lateral pontomedullary syndrome (occlusion of vertebral artery): Combination of lateral medullary and lateral inferior pontine syndromes
5. Basilar artery syndrome (the syndrome of the lone vertebral artery is equivalent): A combination of the various brainstem syndromes plus those arising in the posterior cerebral artery distribution
   Bilateral long tract signs (sensory and motor; cerebellar and peripheral cranial nerve abnormalities): *Bilateral long tract; cerebellar and peripheral cranial nerves*
   Paralysis or weakness of all extremities, plus all bulbar musculature; Cranial

arteries all give rise to 100- to 300-μm diameter branches that penetrate the deep gray and white matter of the cerebrum or brainstem (see Fig. 366-2). Each of these small branches can thrombose either by atherothrombotic disease at its origin or by the development of lipohyalinotic thickening. Thrombosis of these vessels causes small infarcts that are referred to as *lacunes*. They range in size from as small as 3 or 4 mm to 1 or 2 cm. Hypertension is the principal risk factor for such small-vessel disease. Lacunar infarcts cause approximately 20 percent of all strokes.

Clinical manifestations Lacunar infarcts cause recognizable stroke syndromes. Transient symptoms (lacunar TIAs) may herald a lacunar infarct; they may occur several times a day and last only a few minutes. Recovery often begins within hours or days after the infarct, and over weeks or months may be complete or result in minimal residual deficit. In some cases, significant disability persists. The most common lacunar syndromes are the following:

1. Pure motor hemiparesis from an infarct in the posterior limb of the internal capsule, crus cerebri in the midbrain, or basis pontis. The face, arm, leg, foot, and toes are almost always involved.
3. Ataxic hemiparesis from an infarct in the base of the pons.
4. Dysarthria and a clumsy hand or arm due to infarction in the of the pons or in the genu of the internal capsule.
5. Pure motor hemiparesis with "motor aphasia" due to thromb occlusion of a lenticulostriate branch supplying the genu anterior limb of the internal capsule and adjacent white matt the corona radiata.

Syndromes resulting from occlusion of the penetrating arteries o proximal posterior cerebral artery were discussed above. Syndr resulting from occlusion of the penetrating arteries of the basilar a (see Figs. 366-8 to 366-10) include ipsilateral ataxia and crural paresis, pure motor hemiparesis with horizontal gaze palsy, and h paresis with a contralateral sixth nerve palsy. Lower basilar bi syndromes include sudden internuclear ophthalmoplegia, horiz gaze palsy, and appendicular cerebellar ataxia.

An anarthric pseudobulbar syndrome due to bilateral infarc in the internal capsule can occur from disease in the lenticulos arteries. Before the advent of antihypertensive therapy, multip cunes often caused pseudobulbar palsy with emotional instabil slowed abulic state, and bilateral pyramidal signs; this syndro now less common.

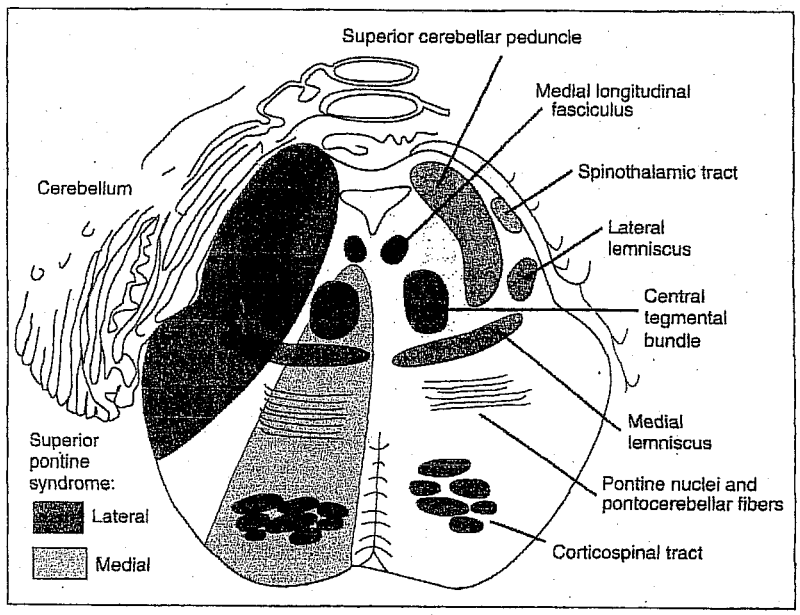


FIGURE 366-8 *(Courtesy of CM Fisher, M.D.)*
Signs and symptoms: *Structures involved*

1. Medial superior pontine syndrome (paramedian branches of upper basilar artery)
   On side of lesion
   Cerebellar ataxia (probably): *Superior and/or middle cerebellar peduncle*
   Internuclear ophthalmoplegia: *Medial longitudinal fasciculus*
   Myoclonic syndrome, palate, pharynx, vocal cords, respiratory apparatus, face, oculomotor apparatus, etc.: *Localization uncertain—central tegmental bundle (?), dentate projection (?), inferior olivary nucleus (?)*
   On side opposite lesion
   Paralysis of face, arm, and leg: *Corticobulbar and corticospinal tract*
   Rarely touch, vibration, and position are affected: *Medial lemniscus*
2. Lateral superior pontine syndrome (syndrome of superior cerebellar artery)
   On side of lesion
   Ataxia of limbs and gait, falling to side of lesion: *Middle and superior cerebellar peduncles, superior surface of cerebellum, dentate nucleus*
   Dizziness, nausea, vomiting; horizontal nystagmus: *Vestibular nucleus*
   Paresis of conjugate gaze (ipsilateral): *Pontine contralateral gaze*
   Skew deviation: *Uncertain*
   Miosis, ptosis, decreased sweating over face (Horner's syndrome): *Descending sympathetic fibers*
   Static tremor reported in one case: *Dentate nucleus (?), superior cerebellar peduncle (?)*
   On side opposite lesion
   Impaired pain and thermal sense on face, limbs, and trunk: *Spinothalamic tract*
   Impaired touch, vibration, and position sense, more in leg than arm (there is a tendency to incongruity of pain and touch deficits): *Medial lemniscus (lateral portion)*

Aortic Atheromatous Disease Atherosclerotic ease of the ascending aorta is a potential source of ce emboli. One study found a strong, independent assoc between atherosclerotic disease of the aortic arch, de strated by transesophageal echocardiography, and th of ischemic stroke. The incidence and natural histo these lesions are uncertain.

LABORATORY AND IMAGING EVA TION Therapy in ischemic stroke is aided by a p diagnosis that determines the primary vascular path and the extent and location of the stroke. The c presentation and temporal profile of a stroke often su its cause. Accurate diagnosis is based largely on the h and examination, supplemented by judicious use of tests and imaging of the brain [CT and MR imaging (and its blood vessels (arterial Doppler ultrasonog and MR and x-ray angiography).

Careful auscultation for bruits of the carotid a and their extracranial branches may add supportir dence for a diagnosis. Ancillary diagnostic studies ally should be used to confirm or exclude other cond rather than to search for evidence of every possible d sis. Chest x-rays, urinalysis, complete blood count, e cyte sedimentation rate, serum electrolytes, blood u trogen, blood sugar, serologic tests for syphilis, seru profile, serum uric acid, blood clotting studies, and t function studies all may be helpful in detecting pre ing causes of vascular thrombosis or intracranial h rhage. An electrocardiogram (ECG) may demonstra duction abnormalities and arrhythmias or reveal ev of recent myocardial infarction. A CT scan will ofte onstrate an area of infarction and will confirm or e the presence of an intracerebral, subdural, or epidur orrhage or other mass lesion. Moreover, it may demc large aneurysms and AVMs and subarachnoid or in tricular blood. A lumbar puncture (LP) will con exclude subarachnoid hemorrhage or meningitis syphilis or other chronic infections. An LP shou be performed on patients with intracranial mass (Chap. 360).

Atherothrombotic Disease of the Internal C Artery and its Branches Several diagnostic tecl are available for evaluating patients with carotid TIA, and stroke. Positive findings must be interpr the appropriate clinical context. For example, a artery bruit in the neck or an ulcerated plaque at th of the internal carotid artery detected by ultrasonc